Dietary Intake of Lead and Blood Lead Concentration in Early Infancy

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 Under circumstances of low prenatal exposure to lead and low nancietary exposure to lead postnatally, four breast-fed infants and 25 formula-fed infants were studied to determine the relation between dietary intake of lead and blood lead concentration. From 8 through 111 days of age, the mean dietary

itake of lead by the formula-led infants was 17 µg/day (3 to 4 µg/kg/day), and intake of lead by the breast-led infants was estimated to be only slightly greater. The mean blood lead concentration at the age of 112 days was \$.1 µg/dL. From 112 through 195 days of age, 17 infants entitlined in the study: ten received a mean dictary intake of lead of 16 µg/day, and seven received a mean intake of 1 µg/day. At 196 days of age, mean blood lead concentrations were significantly different (7.2 and 14.4 µg/dL, respectively).

(Am J Dia Child 1943;137:585-891)

Tesd ingested with food constitutes an important source of environmental lead exposure. Infants are no exception in this regard and may, in fact, be at high risk because of their high level of food intake relative to body weight. A daily permissible intake (DPI) of lead of 300 µg/dsy from

all sources was proposed in 1971 by an ad hoe committee of the Bureau of Community Environment Management, Public Health Service. This value was chosen to preclude an increase in the body ourden of a child between 12 and 30 months of age, and it was based, in part, on the assumption that only 10% of ingested lead, ie, 30 µg/day, would be absorbed. However. results of metabolic balance studles reported in 1972 by Alexander et al' suggested that considerably more than 10% of distary lead, perhans as much as 50% of intake, is absorbed by infants and children.

Based on lead concentrations in foods in 1972, average lead intakes by 6-month-old infants were estimated to be 97 to 119 µg/day. Thus, the quantity of lead absorbed was likely to be greater than 30 µg/day. It therefore seemed desirable to obtain quantitative determinations of lead intake. In addition, we wanted to determine the relationship between distany intake of lead and blood lead concentration, considering blood lead concentration to be a crude index of body burden.

Environmental contamination with lead and, hence, exposure to nondictary sources of lead is relatively low in Iowa City, and these conditions were thought to be favorable for a study of the relationship of dietary intake of lead to body burden of lead. The first

objective of our study was to determine dietary lead intake quantitatively. We had already developed methods for determining the dietary intakes of infants in a quantitative manner for each day of study, and a similar method would permit recording of the dietary intake of lead. The second objective was to measure blood lead concentration and to relate it to dietary intake of lead under conditions of low nondietary exposure.

-The infants studied were enrolled in other studies being carried out in our unit at the time. According to the protocois being followed, from 8 through III days of age, all formula-fed infants consumed products supplied to us in glass-feeding units. Such feedings resulted in low dietary intakes of lead. From 112 through 195 days of age. some of the infants were fed whole cow milk obtained in cartons flow dietary intake of lead), and the other infants were fed milk or formula supplied in cans, resulting in considerably greater intakes of lead. These latter lead intakes were similar to those of many infants being fed commercially available formulas.

These circumstances afforded at opportunity to determine the influence of two levels of dietary intake of lead on blood lead concentration. We believe that the results are relevant to establishment of a new DPI for lead.

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SUBJECTS AND PLAN OF STUDY

Twenty-nine subjects, primarily infants and children of students and farolty at the University of Irwa, were enrolled. All subjeets were white, lived in or near laws City. and were born between August 1975 and January 1976. One set of twins was included. The population of Iowa City at that time was slightly less than 50,000, and support enrollment is the university was approximately 20,000. Birth weights were 2,450 g or more. Four breest-fed and 25 formula-fed infants were enrolled between 6 and 9 days of age. The breast-fed infants were treated as described elsewhere': data concerning these infants are confined to the first 112 days of life. The formula-fed infants made up a subsample of infants participating in various studies of food intake and growth from 8 to 111 days of age. Sevenseen of the formula-fed infants continued their participation from 112 to 195 days of age in a study of diet and gastrointestinal blood loss.

METHODS

The projects were reviewed and approved by the University of Iowa Human Subjects Review Committee. The relevant protocol was explained in detail to one or both parents, and written consent was obtained. The infants visited the Lora N. Thomas Metabolic Unit at our hospital at regular intervals as described in previous publications.

Feedings

A supply of ready-to-feed formula (67 kraidL) was delivered to the family of each of the formula-fed infams until the infant reached III days of age. Deliveries of formula were made weekly. The weight of the bottles delivered for each day was recorded. The empty, partially empty, and mused bottles were collected at the time of the next formula delivery and were weighed. The daily quantity of formula consumed was calculated by subtracting the weight returned from the weight dispensed. From the concentration of lead and the quantity of formula consumed, the intake of lead from formula consumed, the intake of lead from formula consumed.

No attempt was made to determine the quantity of milk communed by the breast-fed infests.

From II2 through ISS days of age, infants were fed milk or formula of three types as described in detail previously; (I) homogenized whole one milk obtained in cartons from a local dairy, (2) a commercially available milk-based formula supplied in quart cana, and (3) homogenized whole one milk supplied in quart cans and heat treated in the same manner as the commercially avail-

able formula. As has been deceribed for bottles, we recorded weights of samens or cars dispensed to and recovered from the families.

Beikost (foods other than milk or formula fed to infants) was delivered to the family from a supply obtained from one manufacturer, and as in the case of formula, the jark were weighed to determine the quantity consumed. Intake of beikost was quantitatively determined for breast-fed and formula-fed infants through 155 days of age. We did not determine the quantity of water consumed but determined the concumulation of lead in water collected in leading amber glass juro from the kitchen top of each home.

Air and Dust

Environmental media other than diet believed to contribute to lead exposure include air and house dust. Air was sampled inside the home and in any other location in which the infant stayed more than 20 house A weeum pump was placed in the room most frequently occupied by the infant. Air was drawn for at least 60 minutes through a mixed cellulose enters (Pillipore) titler (pore size, 0.8 µm) at a rate of 22 LJ min.

Dust was collected by swabbing an apprecimately 1-ft area of a flat horizontal swelves (floor, dresser, table, or television set) with ashless filter paper. All of the samples were obtained before the infants reached 112 days of age.

Blood

An anteculaisal vein was used for obtaining blood from the mothers of the infants. Sleed from an external jugalar vein was obtained from infants within two days of the ages 8, 22, and 56 days and at 28-day intervals (=four days) thereafter. Samples were analyzed for lead within 48 hours or were frozen for subsequent analysis (see "Lead Determination" subsections.

The hemoglobin level was determined as described previously. Free crythrocyte pretoporphyrin (FFP) concentration was determined by the method of Piomelli.

The hemoglobin level was determined by the method of Piomelli.

Lead Determination

Duplicate analyses were made with each of at least three bottles (or cans) of each formula, with several cartons of milk, and with single specimens of human milk or water. These analyses were performed without ashing. In the case of beikost, duplicate analyses were made of at least three jars from each lot. Beikost was mixed with 10% alcoholic magnesium nitrate in porcelain crucibles, dried at 100 °C, and ashed at 410 °C for 12 bours, and the ash dissolved in nitrie acid. Filters containing

dust from air in filter parent values of spefaces were removed with forces from their containers and were ashed as describes for beikost. Single specimens of blood ware prepared by the addition of dilute hiteraacid and outylphenoxypolyethoxyethansi and analyzed without ashing.

Lead determinations were performed by flameiess atomic absorption opening frameters with a graphite formace. A deuterium are lamp was used for benigmound correction. Water and dissolved askes from belicost or filters were read directiv against aqueous standards of lead nutrate. For milk, formula, and blood, the method of standard additions was used, whereby each especimen was read with and without an internal standard. The differences in rendings were averaged for each working day, and this mean value was used to calculate lead concentrations of specimens.

The detection limit was 2 and 1 for the actual determination of lead, Because of saiding and/or diluzion, the innit was 6 and kg for beikest and 20 and for milk, for-nuls, and blood. The esmeentration of load in beikest ranged from less than 1 to 42 and kg.

The within-container variation (technical error) in lead concentration (coefficient of variation) was 7.7% for 12 bottles of formula analyzed in duplicate, 15.4% for nine caus of milk or formula analyzed in duplicate or triplicate, 7.7% for 15 Jers of beikest analyzed in triplicate, and 2.8% for 18 blood specimens analyzed in triplicate.

Accuracy of lead determination was assessed for believs by determining the recovery of addres standard amounts of lead on aine occasions. The mean recovery was 97.9%, with a range of 92% to 196%. Because the method of standard additions was used for milk, formula, and blood, other methods of checking accuracy had to be used. As part of a blinded multilaboratory quality assessment, eight samples were supplied. These consisted of two formulas. each with no added lead, ie. as originally taken from the container, and with three levels of added load (25, 50, and 73 ug/tl). The mean recovery of lead was 100.6% (ED, 17.0%). In the case of blood, 11 specimens were shared with two outside laboratories. With one specimen, values differed appreeighty: 7 mg/dL in our laboratory, 15 mg/dl. in laboratory I, and II mg'dL in laboratory 2. For each of the other ten specimens, our value exceeded that of lab - TI ETT 15 " 0 to 2 world (mean, 1.2 world) and that of laboratery 2 by 2 to 8 ug/dL (mean. 5.7 we dl.).

RESULTS

Twenty-five femoula-fed and four breast-fed infants were studied. The

mean concentration of lead in maternal blood six to nine days after parturation was 9.6 µg/dL (SD, 3.2 µg/dL; range, 4 to 16 µg/dL), suggesting that prenatal lead exposure of the infants had been low.

Dietary intake

The 25 formula-fed infants received a number of Jifferent milk-based and soy protein isolate-based formulas between birth and 112 days of age. The formulas were all supplied ready to feed (67 kml/dL) in gisss buttles, and concentrations of lead ranged from 19 to 26 pg/L, with a mean of 20 µg/L (Table 1). The mean volume of intake of formula from 8 through 111 days of age was 0.783 L/day, so that intake of lead from formula averaged 15 ug/day. The mean total intake (from formula plus belkost) was 17 ug/day (Table I). Data concerning lead intakes by individual infants are given in Table 2. By referring to the subject numbers in Table 2 of this report and the corresponding subject numbers in a previous report, details about the feeding history of each of these 17 infants may be ascer-

From 112 through 195 days of age, 17 formula-fed infants continued in the study. Ten received homogenized whole cow milk supplied in cartons from a local dairy (mean lead concentration, 10 µg/L), four received a commercially available formula supplied in cans (mean lead concentration, 57 µg/L), and three received heattreated cow milk supplied in cans (mean lead concentration, 99 µg/L). In view of the small number of infants receiving milk or formula in cans, we have combined the data concerning - these seven infants. Because distary calcium interferes with absorption of lead," and because the concentration of calcium was considerably greater in the milk supplied in case then in the formula supplied in case (approximately 1.100 v 600 mg/L), the amount of lead absorbed from canned milk may not have been much greater than that absorbed from canned formula. The concentration of lead given in Table I for malk or for crula supplied in cans, ie, 70 μg/L, is the weighted mean calculated by dividing the total amount of ead consumed by the total volume of

			Meen	32 0			
	Ann Rel	11 Days,	Age, 112-18# Days				
	Gless Sottles* (n=25)		Cartons* (n=18)		Cane* (n=7)		
Formuta Volume consumed, L/day	0.783	0.100	0.843	0.159	0.742	0,151	
Louis concentration, ug/L	20		10	•••	70		
Lead intake, ug/day	18	12.5		1.6	5.2	12.9	
Deingal Volume consumed, L/Cay	0.041		0.281		0.305		
Lead concurration, mg/L	32	•	29	•••	30		
Load intake, payday	1.	1.0	8	4.0		4.0	
Total lead intelle	17.	2.5 i	16	3.0	41	15.4	

[&]quot;Container in which milk or formula was supplied.

Subject No.	Age Range, Days									
	9-13	14-27	28-41	42-55	54-43	84-177	112-135	140-187	168-19	
2173	11.0	:5.8	17.1	15.4	164	16.0				
2176	3.0	10.3	10.0	12.4	:2.3	14.8	12.5	14.2	113	
2177	10.3	10.7	10.1	11.7	17.4	23.0	• • • •			
Z196	16.7	23.4	24.8	27.0	22.5	23.1			• • •	
2224	8.6	10.0	11.0	11.4	14.2	21.4	~ 41.5	40.0	57.1	
2225	14.9	16.3	16.1	17.5	17.8	18.8	-53.1	\$2.4	53.4	
2228	13.8	16.0	15.4	16.1	17.4	20.8	-51.5	10.3	55.7	
2229	15.0	17.1	16.4	16.9	19.6	20.8	16.2	13.9	15.1	
223C	11.4	13 1	151	17.8	18.5	18.0	- 43	52.3	44.9	
2231	13.2	13.3	14.7	15.4	15.9	17.8	18.2	17.8	18.0	
2232	13.8	14.6	15.5	17.2	:5.0	21.2	15.4	14.8	15.1	
2233	14.6	19.8	22.3	22.7	21.4	22.0	•••			
2234	14.8	16.5	18.4	17.7	17.9	19.6		•••	•••	
2225	13.1	15.1	16.7	17.5	17.5	18.7	- / -	• • •	•••	
2236	14.5	15.	16.4	15.5	15.0	18.4	••••	•••		
2237	17,4	18.1	:8.6	24.4	28.5	25.2	•••	• • •	•••	
2241	12.4	15.3	15.2	17.5	17.5	19.1	14.3	15.2	17.3	
2243	12.3	13.4	14.8	14.0	15.2	16.8	23.6	28.0	19.0	
2244	11.5	12.3	13.5	13.8	16.8	18.6	16.6	. 20.1	20.9	
2261	12.5	13.7	15.2	15.2	16.1	21,1	~79.2	76.3	20.9 57.4	
2266	8.7	1 7	13.9	15.6	15.8	19.0	15.9	•	•	
2286	13.3	16.3	17.1			_	1	16.5	16.5	
		14.6		17.2	17.6	20.4	15.3	16.1	15.1	
2267	11.5		15.2	14.8	16.1	18.4	- 79.3	63.9	47.8	
	. 13.3	18.1	21.8	25.1	22.5	22.8	- 86.3	85.2	92.8	
2207 Mean	12.6	_ 1 <u>6.0 _</u> 14.8	17.A 15.2	17.2	17.7	19.9	35.2	12.0	13.7	

*Dietary intake of lead is given in micrograms per day throughout.

milk or formula consumed by these seven infants.

As may be seen from Table 1, during the age interval from 112 through 195 days, the mean dietary intake of lead from all sources (milk or formula plus beikost) was 16 <u>ug/day</u> for infants fed milk supplied in carrons and 61 ug/day for infants fed milk or formula supplied in cans. In cal. lating intake, we have ignored the contribution of drinking water because water was not added to

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the fullk or formula before feeding, the lead concentration of water was less than 10 mg/L in all instances, and infants in this age range generally contime only small amounts of drinking

water as such.

Lead concentration in ten samples of human milk collected from the mothers of the four breast-fed infants ranged from 15 to 64 µg/L, with a mean of 25 µg/L. As we reported previously,* these values agree with other values in the literature. Intake of beikost by the breast-fed infants was only slightly less than that by the bottle-fed infants. Thus, assuming that the quantity of human milk consumed by the breastled infants was similar to the quantity of formula consumed by the bottle-fed infants (Table 1), intakes of lead by the four breast-fed infants probably averaged about 20 µg/day. It is possible, however, that because of the lower calcium content, the lead in human milk is better absorbed.

Air and Dust

Air and dust were sampled in the 29 homes of the 23 infants. Lead concentration in air was greater than 0.2 uz/eu m in only a few instances. Values of 6 and 5 µg/cn m were obtained in the home of subject 2233 (Table 2) on two occasions, 19 days. apart. Air was sampled for four hours and in a different room each time.

The estimated air intake at the age of I year is 4 to 6 cu m perday." Assuming 100% retention, lead intake by inhalation of air containing 0.2 µg/cu m would therefore be 0.8 to 1.2 ug/day at the age of I year and presumably no greater at younger ages. Assuming no unusual exposure, eg. industrial. King' estimated that inspired air contributes 2.9% to 5.7% of total lead intake for children aged 12 to 36 months.

Quantities of lead recovered in swabs of dust in the home were less than 8 µg/sq ft in 30 samples from 24 of the 28 homes. Five samples obtained in homes in which three of the infants received day-care supervision also yielded less than 8 µg of lead per squary foot of surface. Swabs obtained in four of the 28 homes yielded greater amounts of lead; two samples were obtained in the home of subject 2175 (Table 2) and yielded 25 ug/sq ft of lead

from a dresser in the subject's bedroom and 8 ug/sq ft of lead from a table in the living room. The sole earnule ob tained from the home of subject 2029 (Table 2) (from a dresser in the subject's bedroom) yielded 14 µg of lead per square foot. In the home of subject 2234 (Table 2), a sample obtained from an antique table, possibly painted at one time with lead paint, yielded a value of 102 µg/sq ft, and a sample obtained from the same table subsequently yielded a value of 85 µg/sq ft. Two other samples were obtained in this home: the amounts of lead were I us/sq ft from a segment of floor and 3 mg/sq ft from a dresser. The only sample obtained in the home of subject 2241 (Table 2) was from a bookcase in the living room that yielded 35 ug/sqft of lead. A similar survey was reported by Sayre et al"; the mean value for lead in 60 samples of suburban household dust was 27 mg/sq ft.

Blood Lead Concentration

Mean concentrations of lead in venous blood at various ages are shown in relation to the type of feeding in the Figure. The mean concert stion of lead in blood of formula-fed infants at the ages of 8, 28, 56, 84, and 112 days (23 to 25 of the 25 infants were represented at each age) were 8.9, 5.8, 5.1, 5.4, and 6.1 µg/dL, respectively. Corresponding SDs were 3.2, 2.2, 1.7, 2.8, and 1.7 ug/dL. Mean blood lead concentrations of the breast-fed infants at these ages were not sharply different from those of the formula-fed infants (Figure). Among the formula-fed infants, the difference in the blood lead concentration between 8 and 56 days of age was statistically significant Kpaired t test, P < .05). Some decrease in concentration would be anticipated because lead in blood is primarily bound to proteins in the erythrocytes. and the physiologic decrease in the erythrocyte count is about 29% from 1 to 2 weeks of age to 6 to 9 weeks of age, with most of the decrease occurring by the age of 28 days." The percent decrease (43%) in the blood lead concentration of the formula-fed infants between 8 and 55 days of age was therefore somewhat greater than that predicted solely on the basis of decrease in the erythrocyte count, probably reflecting encretion from the body or translocation from envision. cytes to other tissues.

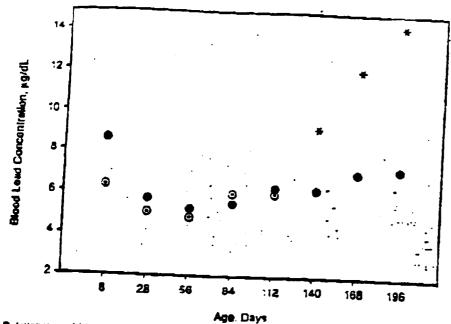
At the ages of 140, 153, and 198 days mean blood lead concentrations of the ten infants whose milk was supplied in cartons (mean lead in ake. 15 µg/day) were 6.2, 7.0, and 7.2 ag dL, respectively (Figure), and SDs were 2.7, 2 g. and 2.7 µg/dL, respectively. Corresponding concentrations of lead in blood of infants who were fed milk supplied in cans (mean lead intake, 61 µg/day) were 9.3, 12.1, and 14.4 µg/dL, and SDs were 4.0, 4.0, and 4.4 μg/dL, respectively. The feedingrelated difference in blood lead concentration was not statistically significant (at the age of 140 days (P = .08) but was significant at the ages of 168 (P<.01) and 196 days (P<.01)

The blood lead concentration of subject 2233 (Table 2), the subject in whose home lead concentration of air was elevated on two occasions—5 and 6 µg/cu m, was 12 µg/dL at the age of 8 days and 12 pig/dL at the age of 28 days (the highest value of any infant as this age), but concentrations at the ages of 56, 84, and 112 days were 6, 4, and 9 μg/dL, respectively. If the values nhexined for lead concentration of air in this infant's home were representative of the entire period from 8 to 112 days of age, it would seem that the daily air intake in the home during the first 112 days of life was appreciably less than the 4 to 6 cu m estimated for 1-year-old infants," and/or that 20 to 36 µg/day of inhaled lead was insufficient to maintain the blood lead concentration above 4 to 9 µg/dL. This infant was not studied after 112 days of

Of the four infants whose homes contained dust with relatively high lead concentrations, an association with an increased blood lead concentration was not detected during the first 196 days of life.

Hemogiobin Level and **FEP** Concentration

Hemoglobin levels and FEP concentrations in relation to feeding are given in Table 3. Neither age-related nor feeding-related differences in levels of hemoglobin were demonstrated. The mean concentration of FEP at the age



Relationship of blood lead concentration to age and dietary imake of lead. Solid circles indicate infants receiving formula supplied in grass bottles (8 through 111 days of age) or milk supplied in cartons (112 through 195 days of age); cosh circles, breast-fed infants; and asterisks, infants receiving milk or formula supplied in cans.

of 196 days was greater (i test, P<.05) for infants who had received milk or formula supplied in cans than for those who had received milk supplied in cari, although the change in FEP contration from 112 to 196 days of age was not significantly different between the two groups of infants.

The greatest blood lead concentrations at 168 and 196 days of age were observed in subjects 2224 (Table 2) (15 and 24 µg/dL, respectively) and 2225 (Table 2) (19 and 18 µg/dL, respectively). These infants also exhibited greater concentrations of FEP at 196 days of age than did any of the other infants (subject 2224, 2.78 µg/g of hemoglobin; subject 2225, 3.42 µg/g of hemoglobin). These elevated values could not be attributed to iron status. Subjest 2224 had values (serum iron level, 48 to 72 µg/dL; transferrin saturation, 12% to 21%) that were only slightly below the mean values for our laboratory, whereas subject 2225 had values (serum iron level, 151 and 103 µg/dL; transferrin saturation, 59% and 36%) that were actually above our mean values.

COMMENT

of mothers of infants in our study

가고 시 : 1980 Am J Dis Child—Vol 137, Sept 1983 was 9.6 up/dL. Although this cuncentration is considerably greater than those reported for adult subjects living in regions remote from industrial activity, and it is less than values commonly reported from industrialized countries. The mean blood lead concentration of 2,646 women from 16 to 75 years of age in the Health and Nutrition Examination Survey II (NHANES II) was 12.8 µg/dL. needs

Thus, prenatal exposure to lead of the infants in our study may be considered low, at least in reference to the general population of the United States. Nondistary exposure to lead from air and dust was also low in most infeats, and distary intake of lead averaged only 17 agiday (Table 1) during the interval from 8 through 111 days of age. With the limited prenatal exposure to lead and limited postnatal exposure to lead from nondietary and distary sources, concentrations of lead in blood were rather stable between 28 and 112 days of age, and the highest mean concentration (at 112 days of age) was only 6.2 ug/dL (Figure). Bluod lead concentrations of ten infants who continued to receive low distary intakes of lead (Table 1, 16 µg/day) increased only slightly between 112 and

Ta	ble :	3.—H	emog	ntrai	Level and tion+	_	
Açe,	!	rdanta MSR F Carto	1967	Infents Fed Milk or Fermula From Gene			
Days	No.	Mear	\$0	No.	Mean SD		
	He	والمشاك	maria e a		264	_	
		121	12	7	12.5 1.1		
140	10	12.8	1.0	7	12.6 1.3		
158	8	13.0	1.0	7	-		
196		12.9			124 0.0		
FEP (Cone		lee	-4- 4			
112	10	223	1.42	7	lenoplobin 1.92 0.35		
140	9	1.93	0.64		1.64 0.34		
168	9	1.79	0.73		171 048		
196	10	1.57	0.43	6	2.32 0.74	i	

FEP indicates free environcyte pressperanytin

196 days of age (Figure). The mean concentration of 7.2 µg/dL at 196 days of age may be contrasted with the average blood lead concentration of 15.0 µg/dL reported for the age interval from 6 months to 2 years in NHANES II."

In contrast with the findings with infants fed diets providing low intakes of lead in the interval from 112 through 195 days of age, a mean lead intake of 11 ugiday during this interval was associated with a statistically significant increase in blood lead concentration. We believe that this report presents the first demonstration that an increase in blood lead concentration can be effected by a lead intake as low as 61 µg/day (8 to 9 µg/kg/day). A significans correlation between dietary intake of lead during approximately the 13th week of life and blood lead concentrations of 13-week-old infants was reported in the Glasgow Duplicate Diet Study. However, it appears from Fig 3 of that report that the significance of the correlation depends on relatively high blood lead concentrations of infants with lead intakes erester than 1.0 mg/wk (143 us/day). Blood lead concentrations less than 16 ug/dL were found in only 22 of 55 infants in the Glasgow study. A duplicate diet study involving 11 infants in Ayr, Scotland," seems to add little to the data available from the Glasgow study:

The elevated concentrations of FEP (2.78 and 3.42 $\mu g/g$ of hemoglobin) at

25-112 tup are 6.2-4/1/

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195 days of age in the two infants (Table 2, subjects 2024 and 2005) with greatest blood lead concentrations (22 and 18 µg/day, respectively) suggests an adverse effect on hemoglobin synthesis associated with an intake of lead of approximately 8 to 9 µg/kg/day for 84 days.

At the present time, there seems to be no major support for the DPI for lead of 300 $\mu \mathrm{g}/\mathrm{day}$ from all sources proposed in 1971. The report by Alexander et al' has been followed by a more extensive report by Ziegier et al,4 and it seems probable that the mean absorption of lead by infants is closer to 40% than to 10% of intake. In 1977, Mahaffey" recommended that the daily intake of lead from all sources be as low

as possible, not to exceed 100 mg day for infants younger than & months of age and not to exceed 150 mg day for older infants and children younger than 2 years of age. The data presented in this report suggest that the body burden of lead of infants increases when dietary intakes of lead are 61 µg/day (Table 1 and Figure). For an approximately 7-kg infant, this intake amounts to 8 or 9 ug/kg/day from diet

The metabolic balance studies reported by Ziegler et al' demonstrated that fecal excretion of lead generally exceeded intake when dietary intake of lead was less than 4 µg/kg/day. The present observations of infants during the interval from 8 through III days of

yage demonstrate that with the horner tary exposure to lead, a mean que and intake of 3 to 4 µg kg day is not associated with an increase in ploud lead concentration. On the other hand, distary blood lead concentrations do increase when distary intakes of lead are 8 to 9 mg/kg/day. These data relate to circumstances in which nondietary intakes of lead seem to have been trivial. Thus, until more data are available, it seems reasonable to set the DPI for lead from all sources closer to 3 than to 8 µg/kg/day.

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